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REPORT NO. 5

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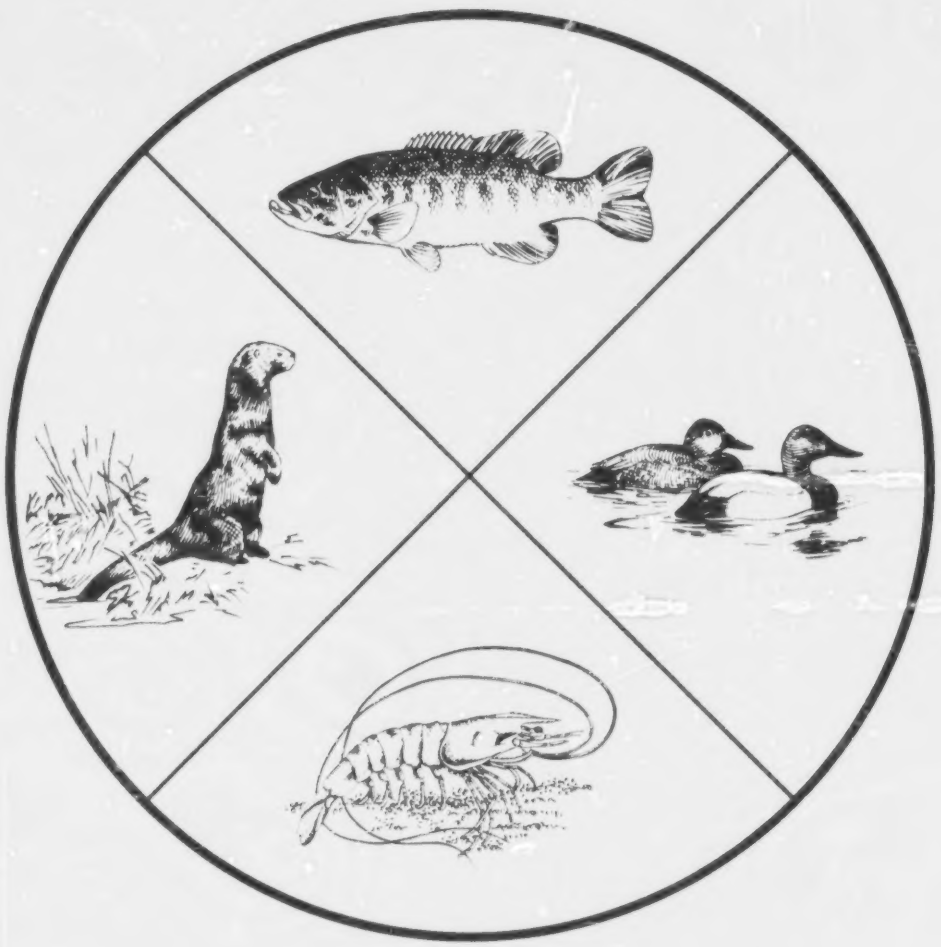
SELENIUM HAZARDS TO FISH, WILDLIFE, AND INVERTEBRATES: A SYNOPTIC REVIEW



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Title	Publication number	Status[a]
Mirex Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review	85(1.1)	Available
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Carbofuron Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review	85(1.3)	Available
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Biological Report 85(1.5)
October 1985

Contaminant Hazard Reviews
Report No. 5

SELENIUM HAZARDS TO FISH, WILDLIFE, AND INVERTEBRATES: A SYNOPTIC REVIEW

by

Ronald Eisler

Patuxent Wildlife Research Center
U. S. Fish and Wildlife Service
Laurel, MD 20708

Fish and Wildlife Service
U.S. Department of the Interior

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SUMMARY

Ecological and toxicological aspects of selenium (Se) in the environment are reviewed, including its chemistry, background residues in biological and other materials, and toxic, sublethal, and latent effects (including the effects of Se deficiency). Recommendations are presented, including proposed Se criteria for protection of sensitive species of fish and wildlife.

Most authorities agree on five points. First, Se deficiency is not as well documented as Se poisoning, but may be equally significant. Second, Se released as a result of anthropogenic activities (including fossil fuel combustion and metal smelting), as well as that in naturally seleniferous areas, poses the greatest threat of poisoning to fish and wildlife. Third, additional research is required on chemical and biological transformations among valence states, allotropic forms, and isomers of Se. Fourth, Se metabolism and degradation are both significantly modified by interaction with various heavy metals, agricultural chemicals, microorganisms, and numerous physicochemical factors, and until these interactions are resolved it will be difficult to meaningfully interpret Se residues in various tissues. And fifth, documented biological responses to Se deficiency or to selenosis vary widely, even among closely related taxonomic groups.

It is generally agreed that Se deficiency may be prevented in fish, small laboratory mammals, and livestock by feeding diets containing 50 to 100 ppb of Se. The concentration range of total inorganic selenite currently recommended for aquatic life protection - 35 ppb in freshwater to 54 ppb in marine waters - is below the range of 60 to 600 ppb that is fatal to sensitive aquatic species. In freshwater, it is also below the range of 47 to 53 ppb associated with growth inhibition of freshwater algae, anemia and reduced hatching in trout, and shifts in species composition of freshwater algal communities. Accordingly, current recommendations for Se with respect to aquatic life appear to afford an adequate measure of protection. However, some studies have shown that Se water concentrations of 9 to 12 ppb are associated with inhibited reproduction of certain freshwater teleosts, suggesting that the current Se criterion for protection of freshwater life should be revised downward. Also, high bioconcentration and accumulation of Se from water by numerous species of algae, fish, and invertebrates is well documented at levels of 0.015 to 3.3 ppb, which are substantially below the recommended range of 35 to 54 ppb. The significance of Se residues in aquatic biota is still unclear, and more research appears to be needed on Se pharmacokinetics

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in aquatic environments.

Aerosol concentrations exceeding 4.0 ug Se/m^3 are considered potentially harmful to human health; however, no comparable data base for birds and other wildlife species is available at this time.

Selenium in natural foods is less toxic than are purified forms of Se. The consensus is that Se toxicity is prevented in livestock if dietary Se concentrations do not exceed 5,000 ppb in natural forage, or 2,000 ppb in feeds supplemented with purified Se. Minimum toxic concentrations of Se in the rat (a sensitive species), fed diets containing natural Se were 1,400 ppb of Se as judged by evidence of liver changes, and 3,000 ppb as estimated from longevity and histopathology; these values were only 350 and 750 ppb, respectively, when diets low in natural Se were fortified with purified Se. The evidence is incomplete for migratory waterfowl and other birds, but diets containing more than 5,000 ppb of Se are demonstrably harmful.

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INTRODUCTION

Selenium (Se) was first identified as an element in 1817 by the Swedish chemist Berzelius. It is now firmly established that Se is beneficial or essential in amounts from trace to part-per-billion concentrations for humans and some plants and animals, but toxic at some concentrations present in the environment. Selenium deficiency was recently reported among cattle grazing in the Florida Everglades, which showed evidence of anemia, slow growth, and reduced fertility (Morris et al. 1984). Conversely, calves of Indian buffaloes died of Se poisoning after eating rice husks grown in naturally seleniferous soils (Prasad et al. 1982); severe reproductive and developmental abnormalities were observed in aquatic birds nesting at Se-contaminated irrigation drainwater ponds in the San Joaquin Valley, California (Ohlendorf et al. in press); and fish contained high body burdens of Se and failed to reproduce in a lake receiving dissolved Se by way of a power plant fly ash sluice water return (Cumbie and Van Horn 1978). Selenium poisoning is an ancient and well-documented disease (Rosenfeld and Beath 1964). Signs of it were reported among domestic livestock by Marco Polo in western China near the borders of Turkestan and Tibet in about the year 1295; among livestock, chickens, and children in Columbia, South America, by Father Pedro Simon in 1560; among human adults in Irapuato, Mexico, in about 1764; and among horses of the U.S. Cavalry in South Dakota in 1857 and again in 1893 (Rosenfeld and Beath 1964). In 1907-08, more than 15,000 sheep died in a region north of Medicine Bow, Wyoming, after grazing on seleniferous plants. The incidents have continued, and the recent technical literature abounds with isolated examples of selenosis among domestic animals.

Selenium has been the subject of many reviews (Rosenfeld and Beath 1964; Frost 1972; Sandholm 1973; Zingaro and Cooper 1974; Frost and Ingvoldstad 1975; Anon. 1975; NAS 1976; Harr 1978; EPA 1980; Lo and Sandi 1980; Shamberger 1981; Wilber 1980, 1983; Fishbein 1977, 1983; NRC 1983; Reddy and Massaro 1983). These authorities agree that Se is widely distributed in nature, being especially abundant with sulfide minerals of various metals, such as iron, lead, and copper. The major source of environmental Se is the weathering of natural rock. The amount of Se entering the atmosphere as a result of anthropogenic activities is estimated to be 3500 metric tons annually, of which most is attributed to combustion of coal; however, aside from highly localized contamination, the contribution of Se by man's activities is small in comparison with that attributable to natural sources. Collectively, all authorities agree that Se may favorably or adversely affect growth, survival, and reproduction of algae and higher plants, bacteria and yeasts, crustaceans,

molluscs, insects, fish, birds, and mammals (including humans). Most acknowledge that sensitivity to Se and its compounds is extremely variable in all classes of organisms and, except for some instances of Se deficiency or of selenosis, metabolic pathways and modes of action are imperfectly understood. For example, selenium "indicator" plants can accumulate Se to concentrations of thousands of parts per million without ill effects; in these plants Se promotes growth, whereas in crop plants accumulations as low as 25 to 50 ppm may be toxic. Thus, plants and waters high in Se are considered potentially hazardous to livestock and to aquatic life and other natural resources in seleniferous zones.

In this account I briefly review the technical literature on ecological and toxicological aspects of selenium (with emphasis on fish and wildlife and their predators and prey), and provide current recommendations for the protection of sensitive species of concern to the U. S. Fish and Wildlife Service. This is part of a continuing series of synoptic reviews prepared in response to informational requests from environmental specialists of the Service.

ENVIRONMENTAL CHEMISTRY

Selenium is characterized by an atomic weight of 78.96, an atomic number of 34, a melting point of 271 C, a boiling point of 685 C, and a density of 4.26-4.79. Chemical properties, uses, and environmental persistence of selenium were documented by a number of researchers whose works constitute the major source material for this section: Rosenfeld and Beath (1964); Bowen (1966); Lakin (1973); Stadtman (1974, 1977); Frost and Ingvoldstad (1975); Chau et al. (1976); Harr (1978); Wilber (1980, 1983); Zieve and Peterson (1981); Robberecht and Von Grieken (1982); Cappon and Smith (1982); and Nriagu and Wong (1983).

There was general agreement on four points. First, that selenium chemistry is complex, and that additional research is warranted on chemical and biochemical transformations among valence states, allotropic forms, and isomers of selenium. Second, that selenium metabolism and degradation is significantly modified by interaction with heavy metals, agricultural chemicals, microorganisms, and a variety of physicochemical factors. Third, that anthropogenic activities (including fossil fuel combustion and metal smelting) and naturally seleniferous areas pose the greatest hazards to fish and wildlife. And fourth, that selenium deficiency is not as well documented as Se poisoning, but may be equally significant. These points are developed here in greater detail.

Selenium chemistry is complex (Rosenfeld and Beath 1964; Harr 1978; Wilber 1983). In nature, selenium exists: as six stable isotopes (Se-74,-76,-77,-78,-80, and -82), of which Se-80 and -78 are the most common, accounting for 50 and 23.5%, respectively; in three allotropic forms; and in five valence states. Changes in the valence state of Se from -2 (hydrogen selenide) through 0 (elemental Se), +2 (selenium dioxide), +4 (selenite), and +6 (selenate) are associated with its geologic distribution, redistribution, and use. Soluble selenates occur in alkaline soils, are slowly reduced to selenites, and are then readily taken up by plants. In drinking water, selenates represent the dominant chemical species. Selenites are less soluble than the corresponding selenates and are easily reduced to elemental Se. In seawater, selenites are the dominant chemical species under some conditions (Cappon and Smith 1981). Selenium dioxide is formed by combustion of elemental Se present in fossil fuels or rubbish. Selenium is the most strongly enriched element in coal, being present as an organoselenium compound, a chelated species, or as an adsorbed element. On combustion of fossil fuels, the sulfur dioxide formed reduces the selenium to elemental Se.

Elemental Se is insoluble and largely unavailable to the biosphere, although it is still capable of satisfying metabolic nutritional requirements. Hydrogen selenide is highly toxic (at 1-4 ppb in air), unstable, acidic, and irritative. Selenides of mercury, silver, copper, and cadmium are very insoluble, although their insolubility may be the basis for the reported detoxification of methylmercury by dietary selenite, and for the decreased heavy metal toxicity associated with selenite. Metallic selenides are thus biologically important in sequestering both Se and heavy metals in a largely unavailable form.

In areas of acid or neutral soils, the amount of biologically available Se should steadily decline. The decline may be accelerated by active agricultural or industrial practices. In dry areas with alkaline soils and oxidizing conditions, elemental Se and selenides in rocks and volcanic soils may oxidize sufficiently to maintain the availability of biologically active selenium. Concentrations of selenium in water are a function of selenium levels in the drainage system and of water pH. In Colorado, for example, streams with pH 6.1- 6.9 usually contain < 1 ppb of Se, but those with pH 7.8-8.2 may contain 270 to 400 ppb (Lakin 1973).

Selenium volatilizes from soils at rates that are modified by temperature, moisture, time, season of year, concentration of water-soluble selenium, and microbiological activity. Conversion of inorganic and organic selenium compounds to volatile selenium compounds (such as dimethyl selenide, dimethyl diselenide, and an unknown compound) by microorganisms has been observed in lake sediments of the Sudbury area of Ontario. This conversion may have been effected by pure cultures of Aeromonas, Flavobacterium, Pseudomonas, or an unidentified fungus, all of which are found in methylated lake sediments. Production of volatile selenium is temperature dependent. Compared with the amount of $(CH_3)_2$ Se produced at an incubation temperature of 20 C, 25% less was produced at 10 C and 90% less at 4 C. Details of Se reduction and oxidation by microorganisms are not clear. One suggested mechanism for selenite reduction in certain microorganisms involves attachment to a carrier protein and transformation from selenite to elemental Se, which in turn may be oxidized to selenite by the action of Bacillus spp., as one example. It is apparent that much additional research on this problem is warranted.

Selenium is an essential nutrient for some plants and animals; it constitutes an integral part of the enzyme glutathione peroxidase and may have a role in other biologically active compounds, especially vitamin E and the enzyme formic dehydrogenase. Some animals require Se-containing amino acids (viz. selenocysteine, selenocystine, selenomethionine, selenocystathionine, Se-methylselenocysteine, and Se-methylselenomethionine), but reportedly are incapable of producing them. Selenium also forms part of certain proteins, including cytochrome C, hemoglobin, myoglobin, myosin, and various ribonucleoproteins. It now appears that selenates and selenites are absorbed by plants, reduced, and then incorporated in amino acids synthesis. The biological availability of Se is higher in plant foods than in foods of animal origin (Lo and Sandi 1980). The net effect of soil, plant, and animal

metabolism is to convert Se to inert and insoluble forms such as elemental Se, metallic selenides, and complexes of selenite with ferric oxides.

Selenium was used in the early 1900's as a pesticide to control plant pests, and is still used sparingly to control pests of greenhouse chrysanthemums and carnations (Rosenfeld and Beath 1964). It has been used to control cotton pests (in Trinidad), mites and spiders that attack citrus, and mites that damage apples. Although no insect-resistant strains have developed, the use of selenium pesticides has been discontinued, owing to their stability in soils and resultant contamination of food crops, their high price, and their proven toxicity to mammals and birds. Selenium shampoos, which contain about 1% selenium sulfide, are still used to control dandruff in humans and dermatitis and mange in dogs. Selenium is used extensively in the manufacture and production of glass, pigments, rubber, metal alloys, textiles, petroleum, medical therapeutic agents, and photographic emulsions.

Air and surface waters generally contain nonhazardous concentrations of Se. Significant increases of Se in specific areas are attributed exclusively to industrial sources, and to leaching of groundwater from seleniferous soils. In the United States, about 4.6 million kg of selenium are released annually into the environment: 33% from combustion of fossil fuels, 59% from industrial losses, and 8% from municipal wastes. Of the total, about 25% is in the form of atmospheric emissions, and the rest in ash. Mining and smelting of copper-nickel ores at Sudbury, Ontario, Canada, alone releases about 2 metric tons of Se to the environment daily, and probably represents the greatest point source of Se release in the world. In 1977, 680,000 kg of Se was produced at Sudbury, but only about 10% was recovered, suggesting that about 90% was lost to the environment. Of the amount lost, perhaps 50 metric tons was dispensed into the atmosphere, probably as selenium dioxide (airborne Se levels 1-3 km from Sudbury were as high as 6.0 ug/m^3). The rest was probably associated with mine tailings, wastewater, and scoria, and is a local source of Se contamination, most notably in lakes. The present annual rates of Se accumulation in lake sediments in the Sudbury area range from 0.3 to 12.0 mg/m^2; these deposition rates exceed those of pre-colonial times by factors of 3 to 18, and are among the highest recorded in North America (Nriagu and Wong 1983).

Selenium is a serious hazard to livestock and probably to people in a wide semiarid belt that extends from inside Canada southward across the United States into Mexico (NRC 1983). Selenium tends to be present in large amounts in areas where the soils have been derived from Cretaceous rocks. Total Se in such soils averages about 5 ppm, but is sometimes as high as 80 ppm. Lack of rainfall has prevented the solution of the Se minerals and the removal of their salts in drainage waters. In some areas, modern fertilization practices and the buildup of sulfates in the soil due to acid precipitation partly lessen the availability of Se to plants and forage crops. In the United States, highly seleniferous natural areas (200-300 ppb in forage) are most abundant in the Rocky Mountain and High Central Plains areas; areas with lower concentrations (20-30 ppb) in forage are typical in the Pacific Northwest and the Southeast. However, huge variations are not uncommon from one specific

location to another. Among plants, primary and secondary selenium accumulators are almost always implicated in cases of acute or chronic selenium poisoning of livestock. Primary Se accumulator plants, such as various species of Astragalus, Oonopsis, Stanelya, Zylorhiza, and Machaeranthera, may require 1 to 50 ppm Se in either soil or water for growth, and may contain 100 to 10,000 ppm Se as a glutamyl dipeptide or selanocystanthionine. Secondary accumulator plants (representative genera: Astor, Gutierrezia, Atriplex, Grindelia, Castillaja, and Comandra) grow in either seleniferous or nonseleniferous soils and may contain 25-100 ppm Se. Nonaccumulator plants growing on seleniferous soils contain 1-25 ppm fresh weight of Se. Meat and eggs of domestic animals may contain 8-9 ppm of Se in seleniferous areas, compared with 0.01-1.0 ppm in nonseleniferous areas. Tissues from animals maintained on high-Se feeds generally contain 3-5 ppm Se fresh weight vs. up to 20 ppm in animals dying of Se poisoning (Harr 1978).

Selenium is nutritionally important as an essential trace element, but is harmful at slightly higher concentrations. Although normal Se dietary levels required to ensure human health range from 0.04 to 0.1 ppm, toxicity may occur if food contains as little as 4.0 ppm. Minimum Se concentrations required are usually higher in livestock than in humans. In areas with highly seleniferous soils, excess Se is adsorbed onto a variety of plants and grains and can be fatal to grazing livestock. There is general agreement, however, that selenium inadequacy can be of greater concern to health than is selenium toxicity. Studies in humans and small animals indicated that Se deficiency (in part) underlies various chronic diseases, increasing susceptibility to cancer, arthritis, hypertension, heart disease, and possibly periodontal disease and cataracts. Selenium reportedly protects mammals and some birds against the toxic effects of mercury, cadmium, arsenic, thallium, and the herbicide paraquat (Hill 1975; Wilber 1983). There is no doubt that high dietary levels of these compounds, as well as of salts of copper, zinc, and silver, contribute to selenium deficiency effects. However, too little is known of the chemical mechanisms to enable scientists to account for these phenomena. Selenium has a comparatively short effectual biological life in various species of organisms for which data are available. Studies with radioselenium-75 indicated that its biological half-life is 10 to 64 days: 10 in pheasants, 13 in guppies and voles, 15 in ants, 27 in eels, 28 in leeches, and 64 in earthworms (Wilber 1983). Many investigators concluded that the greatest current and direct use of selenium is in the transportation of grains grown in seleniferous areas to Se-deficient areas as animal and human food.

BACKGROUND CONCENTRATIONS

Selenium concentrations in nonbiological materials extend over several orders of magnitude (Table 1). In terrestrial materials, concentrations in excess of 5 ppm Se are routinely recorded in meteorites, copper-nickel ores, coal and other fossil fuels, lake sediments in the vicinity of a nickel-copper smeltery, and in sediments of fly ash settling ponds. Water concentrations exceeding 50 ppb Se have been documented in groundwater, especially in areas with seleniferous soils, in sewage wastes, in irrigation drain water, and in water of fly ash settling ponds. Selenium concentrations in air samples were >0.5 ug/m^3 in the vicinity of Se production plants, and these were at least 500 times higher than in a control area. As a result of natural and anthropogenic processes, comparatively high concentrations of selenium in nonbiological materials may pose significant ri-ks to fish and wildlife (this point is discussed later).

Selenium concentrations in representative species of freshwater, marine, and terrestrial flora and fauna are listed in Table 2. Additional information on body and tissue burdens of selenium was given by Birkner (1978), Jenkins (1980), Lo and Sandi (1980), Eisler (1981), and Wilber (1983). It is emphasized that, for all organisms, Se concentrations tended to be significantly higher when collected from locales having certain characteristics: highly seleniferous soils or sediments (de Goeij et al. 1974; Birkner 1978; Speyer 1980; Wilber 1983); high human population densities (Beal 1974); heavy accumulations of Se-laden wastes, such as effluents from systems used to collect fly ash scrubber sludge or bottom ash (Cumbie and Van Horn 1978; Sorensen et al. 1982, 1984); and Se-contaminated subsurface irrigation drainwater (Ohlendorf et al. in press). There is evidence that Se concentrations are positively correlated with mercury residues in piscivorous mammals (Reijnders 1980; Wren 1984) and teleosts (Ganther et al. 1972; Leonzio et al. 1982), although the evidence for teleosts is conflicting (Tamura et al. 1975; Speyer 1980; Maher 1983), and that Se varies seasonally in crustaceans (Zafiropoulos and Grimanis 1977). In general, concentrations of Se in various tissues are usually higher in older than in younger organisms; among marine vertebrates, Se increases were especially pronounced among the older specimens of predatory, long-lived species (Eisler 1984).

All reported Se levels in tissues of marine invertebrates and plants have been less than 2 ppm Se on a wet weight basis, or 12 ppm dry weight. Higher levels are routinely recorded in liver and kidney tissues of marine and coastal vertebrates, including teleosts, birds, and mammals. Livers from

Table 1. Selenium concentrations in nonbiological materials.

Sample, and unit of concentration	Concentration	Reference[a]
TERRESTRIAL (ppm)		
Earth's crust	0.05	Frost and Ingvoldstad 1975
Soils	0.2	Ebens and Shacklette 1982
Limestones	0.08	
Sandstones	up to 0.05	
Shales	0.6	
Chondrites	8.0	
Ocean sediments	0.34-4.8	de Goeij et al. 1974
Coal	3.36(0.46-10.65)	NAS 1976; Kuhn et al. 1980
Fossil fuels	1-10	Harr 1978
Lake sediments		
NY, Lake George	0.22	Heit et al. 1980
Great Lakes	0.35-0.75	Adams and Johnson 1977
Freshwater lakes, Canada	0.2-14.5	Speyer 1980
AQUATIC (ppb)		
Drinking water		
Worldwide	0.12-0.44	Robberecht and Von Grieken 1982
Groundwater		
Nebraska, USA	<1-480	
Argentina	48-67	
Australia	0.008-0.33	
France	<5-75	
Israel	0.9-27	
Italy	<0.002-1.9	
Sewage waters		
USA		
Raw sewage	280	NAS 1976
Primary effluent	45	
Secondary effluent	50	
Worldwide		
Japan	480-700	Ebens and Shacklette 1982
USA	10-280	
USSR	1.8-2.7	
Germany	1.5	
River waters		
Japan	0.03-0.09	Nriagu and Wong 1983
Germany	0.015	Ebens and Shacklette 1982
Amazon River, South America	0.021	
USA		

Table 1. (Continued)

Sample, and unit of concentration	Concentration	Reference[a]
Ohio River	<0.01	Robberecht and Von Grieken 1982
Mississippi River	0.14	
Michigan	0.8-10	
Nebraska	<1-20	
Colorado River	30	
Lake waters		
Sweden	0.04-0.21	Nriagu and Wong 1983
USA	0.04-1.4	NAS 1976
Great Lakes	0.001-0.036	Adams and Johnson 1977
Sea water		
Worldwide	0.009-0.045	Frost and Ingvoldstad 1975; Ebens and Shacklette 1982
Worldwide	0.09-0.45	Whittle et al. 1977
Worldwide	0.09-<6.0	NAS 1976
Israel, Dead Sea	0.8	Robberecht and Von Grieken 1982
Japan	0.04-0.08	
AIR (ug/m^3)		
Near Se industrial plant	0.7	NAS 1976
Control area	0.001	
Near Sudbury (Canada) smelter	up to 6.0	Nriagu and Wong 1983
INTEGRATED STUDIES (ppb)		
California		
Rainwater	0.05	Robberecht and Von Grieken 1982
Lake water	0.018	
Sea water	0.058-0.08	
Irrigation drain water		
Subsurface	300-1,400	Ohlendorf et al. in press
Surface	300	
Vicinity of nickel-copper smelter, Sudbury, Ontario		
Lake waters	0.1-0.4	Nriagu and Wong 1983
Lake sediments	2,000-6,000	
Lake sediments 240 km south of Sudbury	1,000-3,000	
Cu-Ni ores	20,000-80,000	

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Table 1. (Concluded)

Sample, and unit of concentration	Concentration	Reference[a]
Fly ash ponds		
Sediment	14,000	Furr et al. 1979
Water	350	

[a]Each reference applies to the values in the same row, and in the rows that follow for which no other reference is indicated.

Table 2. Selenium concentrations in field populations of selected species of flora and fauna. Values shown are in total Se (mg/kg, or ppm) fresh weight (FW), dry weight (DW), or ash weight (AW). Hyphenated numbers show range and single numbers the mean; where both appear, the range is in parentheses.

Ecosystem, taxonomic group, organism, tissue, location, and other variables	Concentration (ppm)	Reference[a]
MARINE		
Algae and macrophytes		
Whole	0.04-0.24 DW	Chau and Riley 1970; Lunde 1970; Tijoe et al. 1977
Edible seaweeds, whole	0.16-0.39 DW; 0.047 FW	Noda et al. 1979
Molluscs		
American oyster, *Crassostrea virginica*		
Redwood Creek, San Francisco, CA		Okazaki and Panietz 1981
Mantle	4.8 DW	
Digestive gland	8.8 DW	
Kidney	4.7 DW	
Tomales Bay, CA		
Mantle	2.2 DW	
Digestive gland	6.4 DW	
Kidney	2.5 DW	
Transferred from Redwood Creek to Tomales Bay for 56 days		
Mantle	3.5 DW	
Digestive gland	6.5 DW	
Kidney	3.8 DW	
Bivalve molluscs		
Shell	0.03-0.06 DW	Bertine and Goldberg 1972
Soft parts	0.1-0.9 FW; 1.3-9.9 DW	Lunde 1970; Bertine and Goldberg 1972; Karbe et al. 1977; Hall et al. 1978; Fukai et al. 1978; Luten et al. 1980
Muscle	1.1-2.3 DW	Maher 1983
Viscera	1.6-2.5 DW	

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Table 2. (Continued)

Ecosystem, taxonomic group, organism, tissue, location, and other variables	Concentration (ppm)	Reference[a]
Edible flesh, 3 spp.		
Total Se	0.22 (0.16-0.31) FW	Cappon and Smith 1982
As selenate	0.05 FW	
As selenite and selenide	0.17 FW	
Mussel, Mytilus edulis		
Gills	2.0-16.0 DW	Stump et al. 1979
Viscera	up to 5.0 DW	
Mussel, Mytilus galloprovincialis		
Gills	7.0 DW	Fowler and Benayoun 1976c
Soft parts	6.1 DW	
Mantle	5.2 DW	
Viscera	3.2 DW	
Muscle	1.9 DW	
Shell	<0.05 DW	
Echinoderms		
Whole, 7 spp.	0.8-4.4 DW	Papadopoulu et al. 1976
Crustaceans		
Digestive system, 3 spp.	3.0-3.5 DW	Maher 1983
Edible tissues sold for human consumption		
17 spp.	0.2-2.0 FW	Hall et al. 1978; Noda et al. 1979; Luten et al. 1980
Edible tissues, 2 spp.		
Total Se	0.21 FW	Cappon and Smith 1982
As selenate	0.05 FW	
As selenite and selenide	0.16 FW	
Muscle, 5 spp.	2.4-4.4 DW	Maher 1983
Soft tissues, 2 spp.	2.0-2.8 DW	
Copepods, whole	1.8-3.4 DW	Zafiropoulos and Grimamis 1977; Tijoe et al. 1977
Euphausid, Meganyctiphanes norvegica		
Viscera	11.7 DW	Fowler and Benayoun 1976a
Eyes	7.8 DW	
Muscle	1.8 DW	
Exoskeleton	0.8 DW	

Table 2. (Continued)

Ecosystem, taxonomic group, organism, tissue, location, and other variables	Concentration (ppm)	Reference[a]
Shrimp, Lysmata seticaudata		
Viscera	7.0 DW	Fowler and Benayoun 1976c
Eyes	4.8 DW	
Whole	2.6 DW	
Muscle	1.9 DW	
Exoskeleton	1.5 DW	
Molts	0.3 DW	
Sharks		
Muscle	0.2-0.8 FW	Glover 1979
Fish		
Digestive system, 4 spp.	1.0-2.4 DW	Maher 1983
Liver		
2 spp.	2.6-6.6 DW	Grimanis et al. 1978
74 spp.	0.6-5.0 FW	de Goeij et al. 1974; Hall et al. 1978; Luten et al. 1980
13 spp.	5.0-30.0 FW	Hall et al. 1978
Meals, 3 spp.	1.0-4.0 DW	Kifer and Payne 1968
Muscle		
4 spp.	0.5-1.5 DW	Maher 1983
182 spp.	0.1-2.0 FW	Bebbington et al. 1977; Grimanis et al. 1978; Kari and Kauranen 1978; Hall et al. 1978; Noda et al. 1979; Luten et al. 1980
5 spp. Total Se	0.4(0.2-0.6) FW	Cappon and Smith 1982
As selenate	0.1 FW	
As selenite and selenide	0.3 FW	
Whole, 21 spp.	0.3-2.0 FW	Kari and Kauranen 1978; Hall et al. 1978; United Nations 1979
Japanese tunas, 4 spp.		
Liver	10.0-15.0 FW	Tamura et al. 1975
White muscle	0.5-1.3 FW	
Red muscle	3.5-9.1 FW	

Table 2. (Continued)

Ecosystem, taxonomic group, organism, tissue, location, and other variables	Concentration (ppm)	Reference[a]
Black marlin, *Makaira indica*		
Muscle	0.4-4.3 FW	MacKay et al. 1975
Liver	1.4-13.5 FW	
Blue marlin, *Makaira nigricans*		
Kidney	23.0 FW	Schultz and Ito 1979
Blood	1.0 FW	
Gill	1.0 FW	
Muscle		
Total Se	3.3(2.5-4.1) FW	Cappon and Smith 1982
As selenate	0.2(0.09-0.3) FW	
As selenite and selenide	3.1(2.4-3.8) FW	
Striped bass, *Morone saxatilis*		
Muscle	0.3 FW	Heit 1979
Liver	0.6 FW	
Tuna, canned	1.9-2.9 FW	Ganther et al. 1982
Swordfish, *Xiphias gladias*		
Muscle	0.3-1.3 FW	Freeman et al. 1978
Birds		
Kidney, 12 spp.	1.2-5.6 FW	Turner et al. 1978; White et al. 1980
White-faced ibis, *Plegadis chihi*		
Egg	0.3-1.1 FW	King et al. 1980
Royal tern, *Sterna maxima*		
Egg	0.4-2.1 FW	King et al. 1983
Sooty tern, *Sterna fuscata*		
Egg	1.1-1.4 FW	Ohlendorf and Harrison in review
Brown pelican, *Pelecanus occidentalis*		
Egg	0.19-0.38 FW	Blus et al. 1977
Liver	1.0-4.2 FW	
Wedge-tailed shearwater, *Puffinus pacificus*		
Egg	1.1-1.3 FW	Ohlendorf and Harrison in review
Red-footed booby, *Sula sula*		
Egg	0.8-0.9 FW	
Oystercatcher, *Haematopus ostralegus*		

Table 2. (Continued)

Ecosystem, taxonomic group, organism, tissue, location, and other variables	Concentration (ppm)	Reference[a]
Kidney	12.7(2.3-17.5) DW	Hutton 1981
Liver	12.8(5.0-20.5) DW	
Herring gull, *Larus argentatus*		
Kidney	14.1(8.6-19.4) DW	
Liver	7.9(6.9-9.3) DW	
Great skua, *Catharacta skua*		
Kidney	32.8(13.3-89.1) DW	
Liver	19.7(6.7-34.6) DW	
Mammals		
Pilot whale, *Globicephala macrorhynchus*		
Blubber	0.8-1.4 FW	Stoneburner 1978
Liver	22.8-61.6 FW	
Kidney	3.0-10.0 FW	
Ringed seal, *Phoca hispida saimensis*		
Muscle	0.2-2.8 FW	Kari and Kauranen 1978
Liver	29.0-170.0 FW	
Kidney	0.3-3.0 FW	
Blubber	0.06-0.11 FW	
Harbor seal, *Phoca vitulina*		
Juveniles		
Kidney	0.6(0.0-1.3) FW	Reijnders 1980
Liver	2.8(2.6-6.5) FW	
Brain	1.1(0.0-7.4) FW	
Adults		
Kidney	3.5(1.9-7.3) FW	
Liver	109.0(409.0 Max.) FW	
Brain	3.7(1.5-8.2) FW	
Seals		
Liver, 4 spp.	6.1-170.0 FW	Kari and Kauranen 1978; Smith and Armstrong 1978; van de Ven et al. 1979
California sea lion, *Zalophus californianus*		
Mothers with normal pups		
Liver	260.0 DW	Martin et al. 1976
Kidney	22.0 DW	

Table 2. (Continued)

Ecosystem, taxonomic group, organism, tissue, location, and other variables	Concentration (ppm)	Reference[a]
Normal pups		
Liver	4.1 DW	
Kidney	6.1 DW	
Mothers with premature pups		
Liver	79.0 DW	
Kidney	12.1 DW	
Pups born prematurely		
Liver	2.9 DW	
Kidney	3.7 DW	
FRESHWATER		
Algae and higher plants		
Algae, whole	<2.0 DW	Jenkins 1980
Higher plants	0.1 DW	Rossi et al. 1976
Aquatic mosses	0.8 DW	
Filamentous algae		
Se-contaminated area	35.2(12-68) DW	Ohlendorf et al.
Control area	<0.5 DW	in press
Rooted plants		
Se-contaminated area	52.1(18-79) DW	
Control area	0.4 DW	
Molluscs		
Mussels, 3 spp., NY State		
Soft parts	2.0-4.0 DW	Heit et al. 1980
Asiatic clam, Corbicula fluminea		
Whole, Florida	0.7 FW	Winger et al. 1984
Arthropoda		
Zooplankton	0.8-3.9 DW	Adams and Johnson 1977
Plankton		
Se-contaminated area	85(58-124) DW	Ohlendorf et al.
Control Site	2(1.4-2.9) DW	in press
Insects		
Se-contaminated area	20-218 DW	
Control site	1.1-3.0 DW	
Mayfly, Hexagenia sp.	0.3-0.5 FW	Winger et al. 1984

Table 2. (Continued)

Ecosystem, taxonomic group, organism, tissue, location, and other variables	Concentration (ppm)	Reference[a]
Amphibians		
Liver	0.7-4.7 FW	Jenkins 1980
Reptiles		
Water snake, *Natrix* sp.		
Whole, Florida	0.3-0.5 FW	Winger et al. 1984
Fish		
Common carp, *Cyprinus carpio*		
Whole	1.0(0.7-1.4) DW	Wiener et al. 1984
Liver	3.6(2.2-5.2) DW	
Bluegill, *Lepomis macrochirus*		
From water containing <5 ppb Se		
White muscle	0.04 FW	Lemly 1982b
Liver	0.7 FW	
Spleen	1.6 FW	
Erythrocytes	0.04 FW	
Heart	1.0 FW	
From water containing 22.6 ppb Se		
White muscle	3.1 FW	
Liver	11.2 FW	
Spleen	17.7 FW	
Erythrocytes	7.2 FW	
Heart	12.8 FW	
Upper Mississippi River		
Whole	1.2(0.7-1.4) DW	Wiener et al. 1984
Largemouth bass, *Micropterus salmoides*		
From water containing <5 ppb Se		
White muscle	0.05 FW	Lemly 1982b
Liver	0.8 FW	
Spleen	1.8 FW	
Erythrocytes	0.07 FW	
Heart	1.2 FW	
From water containing 22.6 ppb Se		
White muscle	1.7 FW	
Liver	10.2 FW	
Spleen	16.6 FW	
Erythrocytes	8.0 FW	

Table 2. (Continued)

Ecosystem, taxonomic group, organism, tissue, location, and other variables	Concentration (ppm)		Reference[a]
Heart	12.0	FW	
Apalachicola River, Florida			
Whole			
Females	0.3-0.4	FW	Winger et al. 1984
Males	0.3-0.5	FW	
Juveniles	0.3-0.5	FW	
Eggs	0.7-1.0	FW	
Channel catfish, *Ictalurus punctatus*			
Apalachicola River, Florida			
Whole			
Females	1.1-0.3	FW	
Males	0.2-0.3	FW	
Juveniles	0.4-0.6	FW	
Eggs	0.8-2.1	FW	
Threadfin shad, *Dorsoma petenense*			
Whole	0.3-0.5	FW	
Green sunfish, *Lepomis cyanellus*			
From water containing 13 ppb Se			
Liver	7.0-21.4	FW	Sorensen et al. 1984
Muscle	2.3-12.9	FW	
From control site			
Liver	1.3	FW	
Muscle	1.3	FW	
Trout, 2 spp., Wyoming			
From water containing 12.3-13.3 ppb Se			
Liver	50.0-70.0	FW	Kaiser et al. 1979
Muscle	<2.0	FW	
Skin	Max. 4.8	FW	
Mosquitofish, *Gambusia affinis*, whole			
From Se-contaminated irrigation drainwater pond	170(115-283)	DW	Ohlendorf et al. in press
Control site	1.3(1.2-1.4)	DW	
Coho salmon, *Oncorhynchus kisutch*			
Muscle	0.7-1.0	DW	Rancitelli et al. 1968
Liver	3.8	DW	
Chinook salmon, *Oncorhynchus tshawytscha*			
Muscle	1.6	DW	

Table 2. (Continued)

Ecosystem, taxonomic group, organism, tissue, location, and other variables	Concentration (ppm)		Reference[a]
Fish			
Muscle			
25 spp.	0.0-0.5	FW	Uthe and Bligh 1971; Willford 1971; Tong et al. 1971; Pakkala et al. 1972; Rossi et al. 1976; Luten et al. 1980
18 spp.	0.5-1.0	FW	Willford 1971; Tong et al. 1972; Pakkala et al. 1972
3 spp.	1.0-2.0	FW	Schroeder et al. 1970; Pakkala et al. 1972
3 spp.			
Total Se	0.25 FW(0.15-0.34)		Cappon and Smith 1982
As selenate	0.09	FW	
As selenate and selenide	0.16	FW	
10 spp.			
Western Lake Erie	0.4-1.5	FW;	
	1.8-8.1	DW	Adams and Johnson 1977
Liver			
17 spp.	0.0-0.5	FW	Lucas et al. 1970
7 spp.	0.5-1.0	FW	
4 spp.	0.6-5.0	FW	Luten et al. 1980
Whole			
Nationwide, USA			
1972	0.60(0.57-0.64)	FW	May and McKinney 1981
1973	0.46(0.42-0.49)	FW	
1976-77	0.58(0.53-0.62)	FW	
5 spp.	0.5-1.9	FW	Pratt et al. 1972
4 spp.	0.2-0.3	DW	Rossi et al. 1976
6 spp.	0.0-0.5	FW	Walsh et al. 1977
12 spp.	0.5-1.0	FW	
6 spp.	1.0-2.0	FW	
6 spp.	2.1-6.0	FW	
Birds			
Common loon, *Gavia immer*			
Egg	0.4(0.3-0.7)	FW	Haseltine et al. 1983

Table 2. (Continued)

Ecosystem, taxonomic group, organism, tissue, location, and other variables	Concentration (ppm)		Reference[a]
Red-breasted merganser, *Mergus serrator*			
Egg	0.47-1.0	FW	Haseltine et al. 1981
Mallard, *Anas platyrhynchos*			
Egg	0.28-0.81	FW	
From Kesterson National Wildlife Refuge, California, nesting on Se-contaminated irrigation drainwater ponds - 1983			
American coot, *Fulica americana*			
Liver	37.2(21-63)	DW	Ohlendorf et al. in press
Egg	54.0(34-110)	DW	
Ducks, *Anas* spp.			
Liver	28.6(19-43)	DW	
Egg	9.9(2.2-46)	DW	
Black-necked stilt, *Himanoptus mexicanus*			
Egg	32.7(12-74)	DW	
American avocet, *Recurvirostra americana*			
Egg	9.1	DW	
Eared grebe, *Podiceps nigricollis*			
Liver	130.0	DW	
Egg	81.4(72-110)	DW	
From Volta Wildlife Area, California, Control Site - 1983			
American coot			
Liver	5.0(4.4-5.6)	DW	
Ducks, 2 spp.			
Liver	4.1(3.9-4.4)	DW	
Black-necked stilt			
Liver	6.1	DW	
From Apalachicola River, Florida			
Little green heron, *Butorides virescens*			
Whole	0.1-0.5	FW	Winger et al. 1984
TERRESTRIAL			
Fungi	<2.0	DW	Jenkins 1980
Macrophytes			

Table 2. (Continued)

Ecosystem, taxonomic group, organism, tissue, location, and other variables	Concentration (ppm)	Reference[a]
Western wheat grass, Agropyron smithii		
South Dakota, plant top	11.5(0.0-8.4) DW	
Little bluestem, Andropogon scoparius		
Plant top	0.0-6.0 DW	
Asparagus, Asparagus officinale		
Western U.S.	2.7-11.0 DW	
Aster, whole		
Aster caerulescens	560.0 DW	
A. commutatus	Max. 590.0 DW	
A. multiflora	Max. 320.0 DW	
A. occidentalis	284.0 DW	
Milk vetch,		
Astragalus argillosus		
Top	385.0 DW	
Root	27.0 DW	
A. beathii		
Top	1,963.0 DW	
Root	66.0 DW	
A. bisulcatus		
Top	Max. 10,239.0 DW	
Seed	305.1 DW	
A. confertiflorus		
Top	1,372.0 DW	
A. crotulariae		
Top	2,000.0 DW	
Root	45.0 DW	
Locoweed, Astragalus spp.	Max. 46,000.0 AW; Max. 6,000.0 DW	
Saltbush, Atriplex spp.	300.0-1,734.0 DW	
Oats, Avena sativa	2.0-15.0 DW	
Buffalo grass, Bouteloua dactyloides	2.7(0.0-12.0) DW	
Indian paint brush, Castillaja spp.	0.0-1,812.0 DW	
Gumweed, Grindelia squarrosa	38.0(0.0-2,160.0) DW	
Broomweeds,		
Gutierrezia spp.	up to 723.0 DW	
Haplopappus spp.	up to 4,800.0 DW	

Table 2. (Continued)

Ecosystem, taxonomic group, organism, tissue, location, and other variables	Concentration (ppm)	Reference[a]
Tobacco, *Nicotiana tabaccum*		
Leaf	5.8 DW	
Stem	44.2 DW	
Rice, *Oryza sativa*		
Grain	0.09-0.11 FW	
Pear, *Pyrrus communis*		
Fruit	0.02 FW	
Rye, *Secale cereale*	0.9-25.0 DW	
Potato, *Solanum tuberosum*		
Tuber	0.2-0.9 DW	
Wheat, *Triticum aestivum*		
Grain	1.1-35.0 DW	
Stem and leaf	17.0 DW	
Root	36.0 DW	
Corn, *Zea mays*		
Grain	1.0-20.0 DW	
Grape, *Vitis* sp.		
Raisin	<0.001 FW	
Annelids		
Earthworms, whole		
From normal soil	2.2 FW	Birkner 1978
From selenite-enriched soil	7.5 FW	
From soil amended with sewage sludge		
Whole	15.0-22.4 DW	Helmke et al. 1979
Casts	0.6-0.7 DW	
From control field		
Whole	22.1 DW	
Casts	0.6 DW	
Arthropods		
Sow bug, *Porcellio* sp.	0.9 FW	Jenkins 1980
Crane fly, larva, *Tipula* sp.	0.9 FW	
"Fly larvae," whole, from *Astragalus* plant with 1800 ppm Se	20.0 FW	

Table 2. (Continued)

Ecosystem, taxonomic group, organism, tissue, location, and other variables	Concentration (ppm)	Reference[a]
Birds		
Blackbird, Turdus merula		
Whole	2.1 DW	Nielsen and Gissel-Nielsen 1975
House sparrow, Passer domesticus		
Whole	0.6 DW	
Pheasant, Phasianus colchicus		
Whole	0.6 DW	
Mammals		
Beaver, Castor canadensis		
Liver	0.2 FW	Wren 1984
Kidney	0.9 FW	
Intestine	0.04 FW	
Muscle	0.09 FW	
Otter, Lutra canadensis		
Liver	2.1 FW	
Kidney	1.9 FW	
Intestine	1.1 FW	
Muscle	0.2 FW	
Woodchuck, Marmota monax		
From fly ash landfill vicinity		
Adults		
Liver	2.2-10.7 DW	Fleming et al. 1979
Lung	1.4-4.4 DW	
Juveniles		
Liver	3.9-6.4 DW	
Lung	2.1-2.8 DW	
From control area		
Adults		
Liver	0.4 DW	
Lung	0.4 DW	
Juveniles		
Liver	0 .2-0.4 DW	
Lung	0.2 DW	
Field mole, Microtus agrestris		
Whole	0.5 DW	Nielsen and Gissel-Nielsen 1975
White-tailed deer, Odocoileus virginianus		
Muscle	0.16(0.05-0.49) DW	Ullrey et al. 1981

Table 2. (Concluded)

Ecosystem, taxonomic group, organism, tissue, location, and other variables	Concentration (ppm)	Reference[a]
Raccoon, *Procyon lotor*		
Liver	2.8 FW	Wren 1984
Kidney	1.9 FW	
Muscle	0.2 FW	
Rat, *Rattus norvegicus*		
Whole	0.4 DW	Nielsen and Gissel-Nielsen 1975
Rock squirrel, *Spermophilus variegatus*		
Kidney	8.9-53.0 (Max. 90.0) DW	Sharma and Shupe 1977
Mole, *Talpa europaea*		
Whole	2.6 DW	Nielsen and Gissel-Nielsen 1975

[a]Each reference applies to the values in the same row, and in the rows that follow for which no other reference is indicated.

adult seals were comparatively rich in selenium (Table 2); however, high concentrations in liver of maternal California sea lions were not reflected in liver of newborn pups (Martin et al. 1976). Mean concentrations of Se in kidneys of seven species of coastal birds collected from the highly industrialized Corpus Christi, Texas, area usually varied between 1.7 and 5.6 ppm Se fresh weight, but were 10.2 ppm in one bird. According to White et al. (1980), Se concentrations of this magnitude may be sufficient to impair reproduction in shorebirds.

Selenium levels in freshwater biota are relatively low, compared with those in their marine counterparts. In freshwater organisms, about 36% of the total Se was present as selenate, and the rest as selenite and selenide. In marine samples, only 24% of the total Se was present as selenate (Cappon and Smith 1982). The implications of this difference are not now understood, but have great relevance in the ability of Se to complex and detoxify various potentially toxic heavy metals, such as mercury and cadmium. In the most recent Nationwide monitoring of selenium and other contaminants in freshwater fishes, Se ranged from 0.05 to 2.9 ppm (fresh weight, whole fish) and averaged about 0.6 ppm; stations where concentrations in fish exceeded 0.82 ppm (>85th percentile) were in three areas: Atlantic coastal streams; Mississippi River system; and California (May and McKinney 1981). Among fish from Atlantic coastal streams, those from the Delaware River near Camden, New Jersey, had elevated whole body concentrations (i.e., >1.0 and <3.0 ppm Se), which were attributed to the industrialized character of the river. In the Big Horn and Yellowstone Rivers, high Se concentrations in fish may result from geologic sources of the element, including coal, phosphate, and sedimentary rock. Fish from the South Platte River near Denver, Colorado, may receive Se from industrial effluents, or from natural and anthropogenic activities associated with the removal of deposits of coal, barite, and sulfur. In California, where Se was elevated in fish from the San Jouquin River, it was speculated that Selocide, an Se-containing pesticide registered for use on citrus fruits in the 1960's, may have been a source, although contaminated irrigation drain water was considered a more likely possibility (Ohlendorf et al. in press).

Among terrestrial plants, selenium accumulations in species of Aster, Astragalus, and several other genera are sometimes spectacularly high (Table 2). Astragalus is the most widely distributed. About 24 of its more than 200 species are Se accumulators that require Se to grow well. The highest reported Se concentration in plants was 15,000 ppm dry weight, in loco weed, Astragalus racemosus (Wilber 1983). Consumption of these and other Se-accumulating forage plants by livestock has induced illness and death from selenium poisoning. Even at much lower concentrations, Se may harm animals that eat considerable amounts of the forage. Plants that accumulate Se tend to be deeper rooted than the grasses and survive more severe aridity, thus remaining (unfortunately) as the principal forage for grazing in time of drought (Wilber 1983).

DEFICIENCY AND PROTECTIVE EFFECTS

Selenium is a critical nutrient and a key component of several enzymes. There is a general consensus that Se deficiency in livestock is increasing in many countries, resulting in a need for added Se in the food. Selenium deficiency is considered by some researchers to constitute a greater threat to health than selenium poisoning. Studies with animals and humans have suggested that Se deficiency, in part, underlies susceptibility to cancer, arthritis, hypertension, heart disease, and possibly periodontal disease and cataracts (Frost and Ingvoldstad 1975; Shamberger 1981; Robberecht and Von Grieken 1982). These linkages have not yet been demonstrated conclusively; for example, eye lens cataract was induced in 10-day-old male rats by selenate, selenite, selenomethionine, and selenocystine, presumably through interference with glutathione metabolism (Ostadalova and Babicky 1980). On the other hand, adverse effects of Se inadequacy have been clearly documented for a wide variety of organisms, including bacteria, protozoans, Atlantic salmon, rainbow trout, Japanese quail, ducks, poultry, rats, dogs, horses, sheep, swine, cattle, antelopes, gazelles, deer, monkeys, and humans (Jensen 1968; Frost and Ingvoldstad 1975; Jones and Stadtman 1975; Fishbein 1977; Harr 1978; Kaiser et al. 1979; Hilton et al. 1980; Shamberger 1981; Bovee and O'Brien 1982; Robberecht and Von Grieken 1982; NRC 1983; Levander 1983, 1984; Morris et al. 1984). Selenium deficency, whether induced experimentally by use of low Se feeds supplemented with alpha tocopherol or by chronic ingestion of low Se diets, has caused a number of maladies: high embryonic mortality in cattle and sheep; anemia in cattle; poor growth and reproduction in sheep and rats; reduced viabililty of newly hatched quail; nutritional myopathy (white muscle disease) in sheep, swine, and cattle; hepatic necrosis and lameness in dogs, horses, and breeding bulls; hair loss and sterility in rat offspring; and spermatozoan abnormalities in rats. Deficiencies were usually prevented or reversed by supplements with sodium selenate or selenite at 100 ppb Se in the diet, or 20 ug Se/kg body weight administered parenterally.

The availability of Se to plants may be lessened by modern agricultural practices, eventually contributing to Se deficiency in animal consumers. For example, fertilizers containing nitrogen, sulfur, and phosphorus all influence Se uptake by plants through different modes of action, the net effect being a reduction in Se uptake (Frost and Ingvoldstad 1975). The buildup of sulfur (as sulfates) in the soil, due to acid rain, fertilizers, and other sources, interferes with Se accumulation by crops (Frost and Ingvoldstad 1975). In addition, high dietary levels of various heavy metals (including copper, zinc, silver, and mercury) contribute to Se deficiency in animals (Frost and Ingvoldstad 1975; Harr 1978), presumably as a result of Se binding with the metal into biologically unavailable forms (Harr 1978; Kaiser et al. 1979).

The protective action of selenium against the adverse or lethal effects induced by various metals and metalloids is well documented for a wide variety of plant and animal species. Among marine organisms, for example, selenium protects against toxic levels of mercury in algae (Gotsis 1982), shrimp (Lucu and Skreblin 1982), crabs and oysters (Glickstein 1978), fish (Sheline and Schmidt-Nielsen 1977), and mammals (Koeman et al. 1975). Similar observations have been recorded for copper and marine algae (Gotsis 1982); cadmium and freshwater snails (Wilber 1983), marine crabs (Bjerragaard 1982), earthworms (Helmke et al. 1979; Beyer et al. 1982), and rats (Harr 1978); mercury or methylmercury and rats (Cappon and Smith 1982), eggs of lake trout (Klaverkamp et al. 1983b), freshwater teleosts (Kim et al. 1975, 1977) and (temporarily) Japanese quail (El-Bergearmi et al. 1977, 1982; Beijer and Jernelov 1978); and arsenic and freshwater and marine teleosts (Luten et al. 1980; Orvini et al. 1980). Not all tests were conclusive. Studies with some species of freshwater teleosts demonstrated negligible antagonism of Se against mercury (Klaverkamp et al. 1983a) or cadmium (Duncan and Klaverkamp 1983). Selenium reportedly protects mammals and poikilotherms against poisoning by thallium, the herbicide paraquat, cadmium, mercury, lead, arsenic, and copper (Wilber 1983).

Reasons to account for the antagonism of Se and heavy metals (here mercury is used as an example) include dietary source and chemical form of Se, influence of sulfur, biological translocation of Se or mercury to less critical body parts, and chemical linkage of Se to mercury on a linear basis. The exact mode of interaction is probably complex and has not yet been resolved. In regard to diet, Se of animal origin and in the form of selenate is less effective than Se from plant and inorganic sources in preventing methylmercury neurotoxicity in experimental animals (Cappon and Smith 1982). Disruption of sulfur metabolism by Se, the sulfur being replaced by seleno-amino acids and other cell constituents containing Se in living organisms, is one probable cause of Se poisoning. It is conceivable that Se-Hg compounds formed within the organism would be sufficiently nonreactive biologically to interfere with sulfur kinetics, presumably -SH groups (Koeman et al. 1975; Beijer and Jernelov 1978; Cappon and Smith 1982; Gotsis 1982). Differential redistribution of Se or Hg to less critical body parts may partly account for observed antagonisms. Pretreatment of marine minnows with Se protects against Hg poisoning and causes a marked redistribution of Hg among organs, presumably to non-critical body parts, and this transfer may partly account for the observed Se-Hg antagonisms in that species (Sheline and Schmidt-Nielsen 1977). Some investigators have reported that Se results in increased Hg accumulations. Increased retention of Hg and other metals may lead to a higher level of biomagnification in the food chain and higher body burden in the individual, which might counteract the positive effect of decreased intoxication (Beijer and Jernelov 1978). Extensive research is under way on the chemical linkage of Se and Hg. In marine mammals and humans, Se and Hg concentrations are closely related, almost linearly in a 1:1 molar ratio, but this relation blurs in teleosts in which Se is in abundance, and fails in birds (Koeman et al. 1975; Beijer and Jernelov 1978; Orvini et al. 1980; Cappon and Smith 1982).

TOXICITY

MAMMALS AND BIRDS

"The element selenium can be traced in an orderly sequence from its origin in the earth's crust to specific geological formation, to distribution of specific genera and groups of plants which require the element for their growth, to the accumulation in vegetation, and to its subsequent toxicity to birds or mammals that consume the seleniferous foods" (Rosenfeld and Beath 1964). Selenium poisoning in livestock, discussed here, was largely extracted from reviews by Rosenfeld and Beath (1964), Frost (1972), Fishbein (1977), Harr (1978), Shamberger (1981), NRC (1983), and Wilber (1983).

In livestock, there are three basic types of selenium poisoning: acute, resulting from consumption (usually in a single feeding) of a sufficient quantity of highly seleniferous weeds; "blind staggers," from consumption of moderately toxic amounts of seleniferous weeds over an extended period of time; and "alkali disease," caused by the consumption of moderately seleniferous grains and forage grasses over a period of several weeks to months.

Acute poisoning is associated with plant materials containing 400 to 800 ppm Se: sheep died when fed amounts of plant material ranging from 8 to 16 g/kg body weight, or about 3.2 to 12.8 mg Se/kg body weight. The minimum lethal dose of Se administered orally as selenite (mg Se per kg body weight) ranged from 3.3 for horses and mules to 11 for cattle and 15 for swine. Other modes of administration were more toxic, e.g., 2 and 1.2 mg Se/ kg body weight given subcutaneously killed swine in 4 hours and 5 days, respectively; and 1.5-6.0 mg Se/kg body weight given intravenously or intraperitoneally to rats and rabbits were fatal. Accidental toxicosis of sheep and cattle from overtreatment with commercial mixtures of Se salts and vitamin E are also documented for Australia and New Zealand. Acute Se poisoning in domestic livestock is characterized by abnormal movements, lowered head, drooped ears, diarrhea, elevated temperature, rapid pulse, labored breathing, bloating with abdominal pain, increased urination, and dilated pupils. Before death, which is due to respiratory failure, there is complete prostration and lethargy. Duration of illness extends from a few hours to several days, depending on the toxicity of plant material ingested. In these cases, Se is distributed by the circulatory system to all body organs, the concentrations being highest in liver, blood, kidney, spleen and brain, and lowest in muscle, skin, hair and bone. Elimination is primarily in the urine; smaller quantities are excreted

with the feces, breath, perspiration, and bile. Postmortem examinations indicate many pathological changes in the heart, lungs, rumen, liver, kidney, and other organs. No effective treatment is known for counteracting toxic effects of large amounts of ingested selenium.

Chronic selenosis may be induced by dietary exposure to natural selenite, selenate, or seleniferous feedstuffs at dietary concentrations between 1 ppm (rat) and 44 ppm (horse), or from water containing 0.5 to 2.0 ppm of Se. Cattle fed 0.5 mg Se/kg body weight 3 times weekly lost their appetite; sheep fed up to 75 mg selenite daily developed myocardial degeneration and fibrosis, pulmonary congestion, and edema. The minimum toxic concentration of Se in lifetime exposure of rats (a comparatively sensitive species) fed Se-deficient diets fortified with Se was 0.35 mg Se/kg diet, as judged by changes in liver chemistry; and 0.75 mg Se/kg diet, as judged by longevity, and histological changes in heart, kidney, and spleen. These concentrations are 10 times the nutritional threshold for Se, and about 25% of the minimum lifetime exposure to Se in natural feedstuffs that produces similar effects under the same experimental conditions. Signs of chronic selenosis include skin lesions, lymph channel inflammation, loss of hair and nails, anemia, enlarged organs (spleen, pancreas, liver), fatigue, lassitude, and dizziness. "Blind staggers" is characterized by anorexia, emaciation, and sudden collapse, followed by death. Typically, the upper intestinal tract is ulcerated. In "alkali disease," in cattle, hogs, and horses that had eaten seleniferous grains, the signs were deformation and sloughing of the hooves, hair loss, lassitude, erosion of the articular cartilages, reduced conception, increased reabsorption of fetuses, and degeneration of heart, kidney, and liver. It is likely that Se displaces sulfur in keratin, resulting in structural changes in hair, nails, and hooves (Fishbein 1977).

Among birds, it appears that domestic chickens are extremely sensitive to Se; reduced hatching of eggs was recorded at 7 to 9 ppm Se in feed (Ort and Latshaw 1978). Similar results were observed by El-Bergearmi et al. (1977) in Japanese quail at 6 and 12 ppm dietary selenite. Studies in progress at the Patuxent Wildlife Research Center with adult mallards indicated that 100 ppm dietary selenium (as sodium selenite) was fatal within one month, but that survival was high at 25 ppm after 3 months. Poor egg hatchability was recorded in the 25 ppm selenite group, but not in the 10 ppm selenite group; however, hatching percent was reduced in eggs of adults fed 10 ppm of Se as selenomethionine (Dr. G. Heinz, pers. comm.). It appears obvious that additional research is needed on the toxicology of organoselenium compounds.

Selenosis in warm blooded organisms is modified by numerous factors, including method of administration, chemical form of Se, dietary composition, and age and needs of the organism. For example, Se administered in natural feedstuffs is only about one-fourth as toxic as are similar exposures in water or purified feeds (Harr 1978). Concurrent ingestion of minerals and rough or high protein feeds reduces Se toxicity, and exposure by diet is less toxic than exposure parenterally or by inhalation.

Many compounds are known to prevent or reduce toxic effects of subacute

and chronic selenosis in pigs, beef cattle, and other warmblooded organisms. A partial list includes arsenic, strychnine sulfate, tungsten, germanium, antimony, beet pectin, high-fat diets, ACTH injections, sulfate, increased dietary proteins, lactalbumin, ovalbumin, wheat protein, dried brewer's yeast, desiccated liver, linseed oil meal, glucosamine, hemocysteine, creatine, methionine, and choline. Not all of these compounds afforded equal protection against various Se formulations; the reasons for the difference are not clear, but it appears that the subject of selenoprotective agents warrants additional research effort.

AQUATIC ORGANISMS

Among sensitive species of aquatic organisms, death was observed at water concentrations between 60 and 600 ppb Se; early life history stages that were subjected to comparatively lengthy exposures accounted for most of these data (Table 3). Latent mortality after exposure to comparatively high Se concentrations has been documented, but not extensively. For example, all embryos of the zebrafish (Brachydanio rerio) survived exposure to 3,000 ppb of Se during development, but more than 90% of the resultant larvae died soon after hatching; at 1,000 ppb, survival was similar to that in controls (Niimi and LaHam 1975). It has been suggested (EPA 1980) that selenite is more toxic than selenate and is preferentially concentrated over selenate by mussels, Mytilus galloprovincialis (Measures and Burton 1980). Selenite is generally more toxic to early life history stages and effects are most pronounced at elevated temperatures (Klaverkamp et al. 1983a). Also, Se salts may be converted to methylated forms by microorganisms, and these are readily accumulated by aquatic vertebrates (Klaverkamp et al. 1983a). Among freshwater algae species, it has been demonstrated that selenite, selenate, selenomethionine, and selenopurine are all toxic, but that sulphur, as sulphate, has a significant protective role against Se toxicity (Kumar and Prakash 1971). Numerous additional chemical compounds and mixtures probably protect against Se toxicity, much as Se protects against toxic effects of mercury salts and other chemicals, but data are sparse on Se protective agents.

Almost 50 years ago, Ellis et al. (1937) recorded a long list of signs of selenium poisoning in teleosts: loss of equilibrium, lethargy, contraction of dermal chromatophores, loss of coordination, muscle spasms, protruding eyes, swollen abdomen, liver degeneration, reduction in blood hemoglobin and erythrocyte number, and increase in white blood cells. Sorensen et al. (1984), observed most of these signs in Se-poisoned green sunfish together with elevated liver Se concentrations, reduced blood hematocrit, enlarged liver, histopathology of kidney and heart, swollen gill lamellae with extensive cellular vacuolization, and necrotic and degenerating ovarian follicles.

Table 3. Toxicity of selenium salts to aquatic biota. Values shown are in ug/l (ppb) in medium fatal to 50% of the organisms during exposure for various intervals.

Medium, taxonomic group, and species	Exposure interval in hours (h), days (d), or life cycle (LC)	LC-50 (ppb)	Reference[a]
FRESHWATER			
Algae			
Anabaena variabilis	96 h	15,000-17,000	Kumar and Prakash 1971
Anacystis nidulans	96 h	30,000-4,000	
Oedogonium cardiacum	48 h	<100	Nassos et al. 1980
Molluscs			
Snail, *Physa* sp.	96 h	24,000	Reading 1979
Snail, *Physa* sp.	48 h	>10,000	Nassos et al. 1980
Insects			
Mosquito larvae, *Culex fatigans*	48 h	<3,100	
Midge, *Tanytarsus dissimilis*	96 h	42,400	EPA 1980
Crustaceans			
Cladoceran, *Daphnia magna*	48 h	<250	Nassos et al. 1980
"	96 h	710	Halter et al. 1980
"	14 d	430	
"	28 d	240	EPA 1980
"	LC	70-120	
Scud, *Hyallela azteca*	96 h	760	Adams 1976
"	96 h	340	Murphy 1981
	14 d	70	Halter et al. 1980
Cladoceran, *Daphnia pulex*	96 h	3,870	Reading 1979
	LC	600-800	EPA 1980
Amphibians			
Frog, *Xenopus laevis*			

Table 3. (Continued)

Medium, taxonomic group, and species	Exposure interval in hours (h), days (d), or life cycle (LC)	LC-50 (ppb)	Reference[a]
Embryo	27 h	20,000	Browne and Dumont 1979
"	61 h	10,000	
"	96 h	4,000	
"	113 h	2,000	
Tadpole	3 d	8,000	
"	5 d	2,600	
"	7 d	1,500	
Fish			
Goldfish, *Carassius auratus*	96 h	26,100	Cardwell et al. 1976
"	14 d	6,300	
White sucker, *Catostomus commersoni*	48 h	48,600	Duncan and Klaverkamp 1983
	96 h	31,400	
Common carp, *Cyprinus carpio*	24 h	72,000	Sato et al. 1980
	96 h	35,000	Spehar et al. 1982
Northern pike, *Esox lucius*	75 h	11,100	Klaverkamp et al. 1983a
Mosquitofish, *Gambusia affinis*	48 h	>6,000	Nassos et al. 1980
	96 h	12,600	Reading 1979
Channel catfish, *Ictalurus punctatus*	96 h	13,600	Cardwell et al. 1976
Flagfish, *Jordanella floridae*	96 h	6,500	
Bluegill, *Lepomis macrochirus*	96 h	28,500	
"	14 d	12,500	
"	48 d	400-2,000	Adams 1976
Coho salmon, *Oncorhynchus kisutch*			
Fry	43 d	160	
Yellow perch, *Perca flavescens*	10 d	4,800	Klaverkamp et al. 1983a
Fathead minnow, *Pimephales promelas*			
Fry	96 h	2,100	Cardwell et al. 1976
Juvenile	96 h	5,200	
Adult	96 h	2,200-12,500	Adams 1976
"	96 h	620-970	EPA 1980

Table 3. (Continued)

Medium, taxonomic group, and species	Exposure interval in hours (h), days (d), or life cycle (LC)	LC-50 (ppb)	Reference[a]
"	96 h	1,000	Halter et al 1980
"	9 d	2,100	Cardwell et al. 1976
"	48 d	1,100	Adams 1976
	LC	83-153	EPA 1980
Rainbow trout, Salmo gairdneri	96 h	8,100	Spehar et al. 1982
"	96 h	4,200-4,500	Adams 1976
"	96 h	12,500	EPA 1980
"	96 h	7,200-8,800	Hodson et al. 1980
"	9 d	5,400-7,000	
"	21 d	460	Adams 1976
"	96 d	290	
"	LC	60-130	EPA 1980
Brook trout, Salvelinus fontinalis	96 h	10,200	Cardwell et al. 1976
MARINE			
Molluscs			
Pacific oyster, Crassostrea gigas			
Larvae	48 h	>10,000	Glickstein 1978
Crustaceans			
Copepod, Acartia clausi	96 h	1,740	EPA 1980
Copepod, A. tonsa	96 h	800	
Blue crab, Callinectes sapidus	96 h	4,600 (2,700-7,800)	Ward et al. 1981
Dungeness crab, Cancer magister			
Larvae	96 h	1,040	Glickstein 1978
Mysid shrimp, Mysidopsis bahia			
Adult	96 h	1,500 (1,100-2,100)	Ward et al. 1981
Juvenile	96 h	600	EPA 1980

Table 3. (Concluded)

Medium, taxonomic group, and species	Exposure interval in hours (h), days (d), or life cycle (LC)	LC-50 (ppb)	Reference[a]
Egg	LC	127-143	
Brown shrimp, *Penaeus aztecus*	96 h	1,200 (800-1,800)	Ward et al. 1981
Fish			
Fourspine stickleback, *Apeltes quadracus*	96 h	17,350	EPA 1980
Sheepshead minnow, *Cyprinodon variegatus*			
Adult	96 h	7,400-67,100	
Egg through juvenile	LC	470-970	Ward et al. 1981
Pinfish, *Lagodon rhomboides*	96 h	4,400 (2,900-6,700)	
Haddock, *Melanogrammus aeglifinus*			
Larvae	96 h	600	EPA 1980
Atlantic silverside, *Menidia menidia*	96 h	9,725	
Summer flounder, *Paralichthys dentatus*			
Larvae	96 h	3,500	
Winter flounder, *Pseudopleuronectes americanus*			
Larvae	96 h	14,250-15,100	

[a]Each reference applies to the values in the same row, and in the rows that follow for which no other reference is indicated.

SUBLETHAL AND LATENT EFFECTS

Results of laboratory studies and field investigations with fish, mammals, and birds have led to general agreement that elevated concentrations of selenium in diet or water were associated with reproductive abnormalities including congenital malformations, selective bioaccumulation by the organism, and growth retardation. Not as extensively documented, but nevertheless important, are reports of selenium-induced chromosomal aberrations, intestinal lesions, shifts in species composition of freshwater algal communities, swimming impairment of protozoans, and behavioral modifications.

AQUATIC ORGANISMS

In green sunfish from a lake in North Carolina receiving selenium (as fly ash wastes from a coal-fired power station), reproduction failed and the population declined markedly. In these fish, Se levels were elevated in liver (up to 21.4 ppm fresh weight) and other tissues; kidney, heart, liver, and gill showed histopathology; and blood chemistry was altered. Ovaries of fish had numerous necrotic and ruptured egg follicles that may have contributed to the population extinction (Sorensen et al. 1984). It is probable that Se uptake by plankton (containing 41-97 ppm dry weight) from lake water (9-12 ppb) introduced Se to the food chain where it ultimately reached elevated levels in fish through biomagnification (Cumbie and Van Horn 1978). In laboratory tests, however, eggs of common carp hatched normally when incubated in media containing 5,000 ppb of Se (Huckabee and Griffith 1974), as did eggs of lake trout (Salvelinus namaycush) at 10,000 ppb of Se (Klaverkamp et al. 1983b). In frogs (Xenopus laevis), cranial and vertebral deformities and lowered survival were documented during development in water with Se concentrations of 2,000 ppb or higher (Browne and Dumont 1979).

At water concentrations of 47 to 53 ppb, Se was associated with anemia and reduced hatch of rainbow trout (Hodson et al. 1980), growth retardation of freshwater green algae (Hutchinson and Stokes 1975; Klaverkamp et al. 1983a), and shifts in species composition of freshwater algal communities (Patrick 1978). At 250 ppb Se, growth was reduced in rainbow trout fry after exposure for 21 d (Adams 1976), and goldfish demonstrated an avoidance response after 48 h (Weir and Hine 1970). At Se water concentrations of 7,930-11,000 ppb, growth was inhibited in freshwater and marine algae (Patrick et al. 1975; EPA 1978), and swimming rate was reduced in the protozoan

Tetrahymena pyriformis (Bovee and O'Brien 1982). Eggs of channel catfish exposed to certain metals (including cadmium, mercury, and copper) produced an increased percentage of albino fry; however, eggs exposed to 250 ppb Se produced fry with normal pigmentation (Westerman and Birge 1978).

A significant number of chromosomal aberrations was induced in the edible goby, Boleophthalmus dussumieri, by Se after intramuscular and water exposures (Krishnaja and Rege 1982). Intramuscular injections of Se as low as 0.1 mg/kg body weight, or 3,200 ppb in the water column, were associated with a marked enhancement of polyploid cells 76-96 h postadministration; some deaths were recorded at higher test concentrations. Selenite was more effective than selenate in inducing chromosomal aberrations. The authors concluded that a relatively narrow range of Se concentrations lead to a mutagenic rather than a lethal effect.

Accumulation of Se by aquatic organisms is highly variable. In short-term (48 h) laboratory tests at Se water concentrations of 0.015 to 3.3 ppb, Nassos et al. (1980) reported biological concentration factors (BCF) of 460 for mosquitofish to 32,000 for a freshwater gastropod· values were intermediate for daphnids (2,100), plankton (2,600), and Fundulus kansae (3,300), the freshwater killifish. High BCF's (> 680) were recorded for freshwater diatoms subjected to maximum concentrations of 40 ppb Se (Patrick 1978). Livers from rainbow trout and brown trout may contain from 50 to 70 ppm Se fresh weight during lifetime exposure in seleniferous (12.3-13.3 ppb) water, and have BCF values of 3,759 to 5,691 (Kaiser et al. 1979); BCF values were 361 to 390 for skin, and about 180 for muscle (Kaiser et al. 1979). In short-term exposures, most of the Se was probably adsorbed to the body surface (Fowler and Benayoun 1976c), and was then rapidly lost on transfer to Se-free media (Browne and Dumont 1979). In longer exposures, the BCF values in aquatic organisms were lower after immersion in high ambient Se concentrations over extended periods. Thus, marine crabs exposed to a water concentration of 250 ppb of Se for 29 days accumulated Se over water concentration level by a factor of 25 for carapace, and 3.8 for gill; accumulations in muscle and hepatopancreas were negligible. Cadmium in solution enhanced Se uptake (Bjerragaard 1982). Exposure of common carp to 1,000 ppb Se for 85 days resulted in a whole body BCF of 6; additional studies of 7 weeks exposure plus 7 weeks postexposure at Se concentrations between 500 and 5,000 ppb (Sato et al. 1980) yielded a BCF range of 0.6 (5,000 ppb) to 1.8 (500 ppb). Highest BCF's in carp were 50 for kidney and 80 for liver after exposure of the fish to 100 ppb Se for 7 weeks plus 7 weeks in Se-free media. For carp, Se tended to accumulate in kidney, liver, gill, gall bladder, heart, bone, and muscle, in that general order (Sato et al. 1980). Studies with freshwater organisms collected from a farm pond contaminated by fly ash with high Se levels (Furr et al. 1979), and with marine bivalves and nereid worms held for 4 months in seawater flowing through coal fly ash containing 6,200 ppb Se (Ryther et al. 1979), showed that accumulation was slight. Contrasted to this are the observations of Cherry et al. (1976) and Ohlendorf et al. (in press). Cherry and his coworkers collected mosquitofish from a drainage system that received high coal fly ash concentrations at one end and thermal discharges at the other; mosquitofish contained up to 9.0 ppm Se whole body fresh weight. Of 40

elements examined, only Se, zinc, and calcium were accumulated in excess of the levels measured in the water. Ohlendorf et al. found mean residues of 172 ppm dry weight (range 110-280 ppm) in whole mosquitofish from irrigation drainwater ponds contaminated by about 300 ppb of Se; based on a wet/dry factor of 4, the BCF for whole mosquitofish was >91.

Selenium accumulation is modified by water temperature, age of the organism, organ or tissue specificity , mode of administration, and other factors. In the marine mussel, Mytilus galloprovincialis, an increase in water temperature from 13 to 29 C doubled the BCF in 13 days (Fowler and Benayoun 1976b). Mussels preferentially accumulated selenite over selenate (Fowler and Benayoun 1976b); however, mussels did not reach a steady state in 63 days (Fowler and Benayoun 1976c), indicating that Se kinetics in some species are difficult to elucidate in short-term studies. Accumulation rates were higher in small than in large mussels (Fowler and Benayoun 1976b), as they were in freshwater teleosts (Furr et al. 1979). However, the reverse was documented for marine mammals and teleosts (Eisler 1984). When Se was available from both the diet and the medium, concentrations were highest in liver, kidney, and gills of teleosts (Sorensen et al. 1982; Furr et al. 1979; Kaiser et al. 1979), exoskeleton of crustaceans (Fowler and Benayoun 1976c; Bjerragaard 1982), and visceral mass and gills of molluscs (Fowler and Benayoun 1976c). When Se was administered in food to marine shrimps, concentrations were highest in viscera and exoskeleton, suggesting that ingested Se is readily translocated from internal to external tissues (Fowler and Benayoun 1976c). Concentrations of Se in crustaceans usually were higher in fecal pellets than in the diet; fecal pellets may represent a possible biological mechanism for downward vertical transport of Se in the sea (Fowler and Benayoun 1976a), as well as in freshwater environments.

The time for 50% excretion of accumulated Se has ranged from 13 to 181 days in various species of marine and freshwater fauna. Biological half-life of Se accumulated from the medium has been estimated at 28 days for carp (Sato et al. 1980), 37 days for the marine euphausid crustacean Meganctiphanes norvegica (Fowler and Benayoun 1976a), 63 to 81 days for the marine mussel Mytilus galloprovincialis (Fowler and Benayoun 1976b), 58 to 60 days for the marine shrimp Lysmata caudata (Fowler and Benayoun 1976b), and, as reviewed by Stadtman (1974, 1977), 13 days for guppies, 27 days for eels, and 28 days for leeches. Studies by Lemly (1982a) with bluegills and largemouth bass showed elevated tissue levels after exposure to 10 ppb of Se for 120 days. Time for 50% excretion in 30-day elimination trials was about 15 days from gill and erythrocytes; however, there was essentially no elimination from spleen, liver, kidney, or muscle. It appears that research is needed on preferential tissue retention of Se and its implications for biochemical and metabolic transport mechanisms.

TERRESTRIAL INVERTEBRATES

Concentrations of Se decreased in whole earthworms from 22.4 to 15.0 ppm (dry weight) as the rate of sludge application increased from 15 to 60 metric tons/ha. Concentrations of Se in soil and sludge were 0.3 and 0.5 ppm dry weight, respectively (Helmke et al. 1979). Other studies indicated that some metals, notably cadmium, decreased in worms living in soils amended with sewage sludge but that Se concentrations were not affected (Beyer et al. 1982). The biological half life of Se in earthworms is estimated to be 64 days, a period consistent with values of 10 to 81 days documented for ants, birds, mammals, and aquatic biota (Stadtman 1974, 1977).

BIRDS

Embryos of the domestic chicken (Gallus domesticus) are extremely sensitive to Se. The hatchability of eggs is reduced by concentrations of Se in feeds (6 to 9 ppm) that were too low to produce poisoning in other avian species. Dietary Se excess was associated with decreased egg weight, decreased egg producton and hatchability, anemia, elevated kidney Se residues in chicks, and a high incidence of grossly deformed embryos with missing or distorted eyes, beaks, wings and feet (Ort and Latshaw 1978; Harr 1979). Selenomethionine was more effective than sodium selenite in raising the Se content of tissues and eggs (Moksnes 1983). Until recently, there was little credible evidence that selenium adversely affected wild birds. However, Ohlendorf et al. (in press) reported severe reproductive effects in ducks (Anas spp.), American coot (Fulica americana), and other species of aquatic birds nesting at irrigation drainwater ponds in the San Joaquin Valley, California. Water in these ponds contained abnormally high concentrations of about 300 ppb of Se, but low or non-detectable levels of silver, chromium, arsenic, cadmium, mercury, lead, and zinc. Of 347 nests examined from this site, about 40% had at least one dead embryo and about 20% had at least one embryo or chick with obvious external anomalies, including missing or abnormal beaks, eyes, wings, legs, or feet. In addition, brain, heart, liver, and skeletal anomalies were recorded. Concentrations of selenium (ppm, dry weight) were 2-110 in eggs and 19-130 in livers of birds, 12-79 in plants, 23-200 in invertebrates, and 110-280 in fish from the ponds, or 7 to 130 times those found at a nearby control area. It was concluded that Se was the probable cause of poor reproduction and developmental abnormalities in the aquatic nesting birds, due to interference with their reproductive processes. The concentrations of Se in breast muscle of coots were sufficiently high (up to 11.0 ppm fresh weight) to induce State agencies to post the area, advising against the consumption of more than one meal per week of this species, or of any coots by children or pregnant women (Ohlendorf et al. in press).

In studies at the Patuxent Wildlife Research Center, Dr. G. Heinz (pers. comm.) is evaluating effects of dietary Se on mallard reproduction. Ducks given diets containing 100 ppm of inorganic selenite usually died within a month; for some time before death they exhibited reduced food intakes

(bordering on repellence) and significant weight loss. In birds receiving 25 ppm, survival was high after 3 months, but growth and reproduction were dramatically reduced. Teratogenic effects were evident at 3 months in ducklings of mallards receiving 10 ppm, and preliminary evidence suggested that selenomethionine at 10 ppm dietary Se was more effective than inorganic selenite in producing developmental abnormalities. Ducks and their progeny appeared normal after 3 months of exposure to 5 ppm and less of dietary selenite.

MAMMALS

Harr (1978) and NRC (1983) summarized nonlethal effects of selenium on mammals, including reproductive anomalies. Selenosis caused congenital malformations in rats, mice, swine, and cattle. In general, young born to females with selenosis were emaciated and unable to nurse. Mice given Se in drinking water reproduced normally for three generations, but litters were fewer and smaller when compared to controls, pups were runts with high mortality before weaning, and most survivors were infertile.

In rats, selenium did not induce cirrhosis or neoplasia; however, intestinal lesions were observed among those fed diets containing 0.8 to 1.0 ppm Se during lifetime exposure. The threshold requirement for optimal rat nutrition under similar conditions is about 0.08 ppm, again demonstrating the relatively narrow range separating selenium deficiency from selenium poisoning. Absorption of oral radioselenite by rats was as high as 95 to 100%. A single dose of radioselenite concentrated in the pancreas, intestine, erythrocytes, liver, kidney and testes, in that general order; tissue distributions from chronic exposure were similar. As expected, levels of Se in poisoned rats were highest in liver and kidney. Rats, and probably other mammals, can regulate dietary Se accumulations. Dietary concentrations in excess of 54-84 ppb were usually excreted in urine; however, when Se intake exceeded 1000 ppb, pulmonary excretion was active. Excretion of Se in feces, bile, saliva, and hair appears to be relatively constant regardless of the amount of exposure. Yonemoto et al. (1983) demonstrated that some selenotoxic effects in mice, including abortion and maternal death, were prevented by prior treatment with vitamin E, but exacerbated by reduced glutathione; the mechanisms for these interactions are unknown, and merit additional research.

CURRENT RECOMMENDATIONS

All investigators appear to agree on four points. First, that insufficient selenium in the diet may have harmful and sometimes fatal consequences. Second, that exposure to grossly elevated levels of Se in the diet or water, is inevitably fatal over time to terrestrial and aquatic organisms. Third, that there is a comparatively narrow concentration range separating effects of selenium deficiency from those of selenosis. And fourth, that additional fundamental and basic research is required on selenium metabolism, physiology, recycling, interactions with other compounds or formulations, and chemical speciation in order to elucidate its nutrititive role as well as its toxic effects. Accordingly, the proposed selenium criteria shown in Table 4 for prevention of Se deficiency and for protection of aquatic life, livestock, crops, and human health, should be viewed as guidelines, pending acquisition of additional, more definitive, data.

Regarding Se deficiency, it appears that diets containing 50 to 100 ppb Se provide adequate protection to humans and to various species of fish, small laboratory mammals, and livestock (Table 4). Factors contributing to Se deficiency in crops include increasing use of agricultural fertilizers and increasing atmospheric fallout of sulfur; furthermore, foliar applications of selenate, although efficient in raising Se levels in plants, have only short-term value (Frost and Ingvoldstad 1975). There is a general consensus that Se deficiency in livestock in many countries is increasing, resulting in a need for added Se in the food chain.

Current recommendations for protection of freshwater aquatic life are that inorganic selenite concentrations in the water should not exceed 35 ppb on a daily average, or 260 ppb at any time (Table 4). These values are higher for saltwater life: 54 ppb daily average, 410 ppb at any time. The concentration range of 35 to 54 ppb recommended for protection of aquatic life is below the range of 60 to 600 ppb that is fatal to various sensitive species of marine and freshwater fauna, and in this respect affords an adequate measure of protection. It is very near the range (47 to 53 ppb) that has been associated with growth inhibition of freshwater algae, anemia and reduced hatch in rainbow trout, and shifts in species composition of freshwater algal communities. But studies by Cumbie and Van Horn (1978) and Sorensen et al. (1984) showed that water concentrations of 9 to 12 ppb of Se were associated with reduced reproduction of freshwater fishes, and their results strongly

Table 4. Proposed selenium criteria for prevention of Se deficiency and for protection against selenosis.

Criterion	Selenium concentration	Reference
PREVENTION OF SE DEFICIENCY		
Rainbow trout, *Salmo gairdneri* (water levels of 0.4 ppb Se)	70 ppb in diet	Hilton et al. 1980
Poultry	30-50 ppb in diet	Fishbein 1977
Rats	54-84 ppb in diet	Harr 1978
Cattle	20 ppb in diet	Fishbein 1977
Grazing sheep and cattle	100 ppb in diet	Shamberger 1981
Humans	40-100 ppb in diet	Wilber 1983
"	50-200 ppb in diet	Harr 1978
"	60 ug daily (range 22-220)	Robberecht and Von Greichen 1982
"	100-200 ug daily	Shamberger 1981
"	20 ppb in drinking water (if water sole Se source, 2 liters/day)	Robberecht and Von Greichen 1982
PROTECTION AGAINST SELENOSIS		
Aquatic life protection		
Freshwater		
As inorganic selenite	Up to 35 ppb in water (24 hr average), not to exceed 260 ppb at any time	EPA 1980
As inorganic selenate	<760 ppb	EPA 1980
Great Lakes	10 ppb in water	Wong et al. 1978
Saltwater, as inorganic selenite	Up to 54 ppb in water (24 hr average), not to exceed 410 ppb at any time	EPA 1980
Livestock protection		
Drinking water	<50 ppb	NAS 1974; Wilber 1983

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Table 4. (concluded)

Criterion	Selenium concentration	Reference
Diet (total)	<2,000 ppb (dry wt.)	NRC 1983
Diet (natural)	<4,000 ppb	Wilber 1983
Feeds (natural)	<2,000 ppb	Frost 1972
Forage (natural)	<5,000 ppb	Frost 1972
Crop protection		
Irrigation water	<50 ppb	Birkner 1978; Wilber 1983
Human health protection		
Seafood	Not to exceed 2,000 ppb fresh wt.	Bebbington et al. 1977
Drinking water	<500 ppb (as sole Se source)	Robberecht and Von Greichen 1982
Food (natural)	<5,000 ppb	Wilber 1983
Milk or water	<500 ppb	Wilber 1983
Daily intake (all sources)		
Adults		
Safe	200 ug	Shamberger 1981
Normal	60-250 ug	Lo and Sandi 1980
Maximum tolerable level	<500 ug	Levander 1984
Infants	4-35 ug	Lo and Sandi 1980
Children		
Age 1-3	20-80 ug	Levander 1984
Age 4-6	30-120 ug	Levander 1984
Age 7-11 +	50-200 ug	Levander 1984
Air		
Japan	<100 ug/m^3	NAS 1976
USSR	<100 ug/m^3	NAS 1976
USA	<200 ug/m^3	NAS 1976
-	1 to 4 ug/m^3	Harr 1978

indicated that some downward modification of the Se freshwater aquatic life protection criterion may be appropriate. Furthermore, high bioconcentration and accumulation of Se from the water column by numerous species of algae, fish, and invertebrates is well documented at levels between 0.015 and 3.3 ppb, which is substantially below the recommended range of 35 to 54 ppb. The significance of Se residues in aquatic biota in terms of bioavailability and Se receptor sites is imperfectly understood, and it appears that much additional research is warranted on Se pharmacokinetics in aquatic environments.

Livestock appear to be protected against selenosis provided that their diets contain less than 4,000 ppb of natural (i.e., nonsupplemented) selenium (Table 4). This concentration is somewhat higher than levels reported for rats (Wilber 1983). Minimum toxic concentrations of Se in lifetime exposure of rats given diets containing natural Se were 1,400 ppb dietary Se, as judged by evidence of liver changes, and 3,000 ppb as estimated from longevity and histological changes in heart, kidney, and spleen. These values were only 350 and 750 ppb, respectively, when rat diets contained purified, rather than natural, Se. This relationship emphasizes that accidental poisoning of livestock, and presumably fish and wildlife, may occur when soils are deliberately supplemented with purified Se, or when soils or aquifers are contaminated as a result of faulty waste disposal practices. Athough the concentration of < 50 ppb for livestock drinking water and irrigation water for crop protection is inconsistent with that of < 35 ppb for aquatic life protection, neither livestock nor crops appear threatened at the higher level. Since many waterways that abut agricultural lands or areas of high anthropogenic loadings of Se contain valuable and desirable aquatic species, it would appear that some downward modification of the current livestock drinking water concentration of Se is necessary.

Recent field studies demonstrated that migratory waterfowl were heavily and adversely affected while nesting at Se-contaminated irrigation drainwater ponds in California, where food chain organisms contained between 12,000 and 280,000 ppb of Se. The source and fate of Se in irrigation drainwater ponds are largely unknown; they must be determined so that alternate technologies for Se control can be implemented to protect waterfowl in that geographical region.

Aerosol concentrations in excess of 4.0 ug Se/m^3 are potentially harmful to human health (Harr 1978); concentrations in excess of this value (6.0 ug Se/m^3) were regularly encountered in the vicinity of the smeltery at Sudbury, Canada (Nriagu and Wong 1983). It is not now known whether respiration rates of wildlife, particularly birds, are comparable to those of humans, or whether Se absorption energetics are similar, or whether wildlife species that frequent point sources of air contaminated by high Se levels for protracted periods are at greater risk than humans. Until additional and more conclusive data become available, aerosol concentrations of less than 4.0 ug Se/m^3 are recommended for the protection of sensitive species of wildlife.

Acute lethal doses for livestock species ranged from 3,300 ug/kg body

weight of Se for horses and mules to 15,000 ug/kg for swine; appetite loss in cattle was noted at 500 ug/kg. For humans, the maximum tolerance level is usually set at 500 ug of Se daily (Lo and Sandi 1980), and the "safe" level at 200 ug/day (Shamberger 1981). Selenium dietary levels for man should not exceed 5,000 ppb; however, recommended maximum dietary levels in other mammals ranged from 1,000 ppb for rats to 4,000 ppb for horses. For all species, including man, there is a tendency to list Se dosage levels in terms of "natural" and "supplemented" levels, with the tacit understanding that "natural" levels are about one-fourth as toxic as "supplemented" values. Given the complexities of Se metabolism and speciation, it appears that greater precision and clarity are necessary in formulating Se criteria if these criteria are to become administratively enforced standards through passage of appropriate legislation.

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Ecological and toxicological aspects of selenium (Se) in the environment are reviewed, including its chemistry, background residues in biological and other materials, and toxic, sublethal, and latent effects (including the effects of Se deficiency). Current recommendations are presented, including proposed Se criteria for protection of sensitive species of fish and wildlife.

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Lloyd Five Hundred Building, Suite 1692
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